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www.jem.org**Insertion of phosphoglycerine kinase (PGK)-neo 5' of Jlambda1 dramatically enhances VJlambda1 rearrangement.****Sun T, Storb U.**

Department of Molecular Genetics and Cell Biology, University of Chicago, 58th St., Chicago, IL 60637, USA.

Gene-targeted mice were generated with a loxP-neomycin resistance gene (n) cassette inserted upstream of the Jlambda1 region and replacement of the gly codon in the Clambda1 gene with a serine codon. This insertion dramatically increases Vlambda1-Jlambda1 recombination. Jlambda1 germline transcript in pre-B cells and thymus cells are also greatly increased, apparently due to the housekeeping phosphoglycerine kinase (PGK) promoter driving the neo gene. In contrast, deletion of the neo gene causes a significant decrease in VJlambda1 recombination to levels below those in normal mice. This reduction is due to a site left on the chromosome which reduces the Jlambda1 germline transcript. Thus, the correlation between germline transcription and variable (V), diversity and joining (J) recombination is not just an all or none phenomenon. Rather, transcription efficiency is directly associated with the recombination efficiency. Furthermore, Jlambda1 and Vlambda1 germline transcription itself is not sufficient to lead to VJ recombination in T cells or early pre-B cells. The findings may suggest in vivo: (a) locus and cell type-specific transactivators direct the immunoglobulin cell receptor loci, respectively, to a "recombination factory" in the nucleus, and transcription complexes deliver V(D)J recombinase to the recombination signal sequences.

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ajpcell.physiology.org**LEDGF regulation of alcohol and aldehyde dehydrogenases in lens epithelial cells: stimulation of retinoic acid production and protection from ethanol toxicity.****Fatma N, Kubo E, Chylack LT Jr, Shinohara T, Akagi Y, Singh DP.**

Department of Ophthalmology, 985840 Nebraska Medical Center, University of Nebraska Medical Center, Omaha, NE 68198-5840, USA.

Retinoic acid (RA) is required for the normal growth and maintenance of many cell types, including lens epithelial cells (LECs). Alcohol (ADH) and aldehyde (ALDH) dehydrogenases are implicated in cellular detoxification and conversion of vitamin A to RA. Lens epithelium-derived growth factor (LEDGF) provides cellular protection against stress by transactivating stress-associated genes. Here we show evidence that LEDGF binds and transactivates heat shock (nGAAn) and stress response (A/TGGGGA/T) elements in the promoters of ADH1, ADH4, and retinaldehyde (RALDH2) genes. Electrophoretic mobility and supershift assays disclosed specific binding of LEDGF to nGAAn and A/TGGGGA/T elements in these gene promoters. Transfection experiments in LECs with promoters linked to a chloramphenicol acetyltransferase (CAT) reporter gene along with LEDGF cDNA revealed high CAT activity. RT-PCR results confirmed that LECs overexpressing LEDGF contained increased levels of ADH1, ADH4, and RALDH2 mRNA. Notably, LECs displayed higher LEDGF mRNA and protein expression during ethanol stress. Overexpressing LEDGF typically exhibited elevated RA levels and survived better during ethanol stress. The present findings indicate that LEDGF is one of the transcriptional activators of these genes that facilitates cellular protection against ethanol stress and plays a role in RA production.

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